```
=> d his ful

     FILE 'REGISTRY' ENTERED AT 14:09:48 ON 03 SEP 2004
                E DMSO/CN
L12             1 SEA ABB=ON  DMSO/CN
                E SEROTYPE 3 VIRUS/CN

     FILE 'HCAPLUS' ENTERED AT 14:10:12 ON 03 SEP 2004
L13           894 SEA ABB=ON  ?CELL?(W)?COMP? AND ?TRANSPLANT?
L14           105 SEA ABB=ON  L13 AND (?REOVIRUS? OR ?REOVIRIDAE? OR ?VIRUS?)
L15             0 SEA ABB=ON  L14 AND (?ONCOLYS?(3A)RAS?(W)?MEDIAT? OR ?RASMEDIAT
                ?)
L16             7 SEA ABB=ON  L14 AND RAS?
L17             0 SEA ABB=ON  L14 AND ?ONCOLYS?
L18             7 SEA ABB=ON  L14 AND ?AUTOLOG?
L19            92 SEA ABB=ON  L14 AND (?MAMMAL? OR ?ANIMAL? OR ?AVIAN? OR ?BIRD?
                OR ?HUMAN? OR ?SEROTYP?(W)3 OR ?DEARING?(W)?STRAIN?)
L20            94 SEA ABB=ON  L16 OR L18 OR L19
L21             0 SEA ABB=ON  L20 AND (?ANTI?(W)?REOVIRUS? OR ?ANTIREOVIRUS?)(W)?
                ANTIBOD?
L22             0 SEA ABB=ON  L20 AND (?ANTI?(W)?REOVIRUS? OR ?ANTIREOVIRUS?)
L23             1 SEA ABB=ON  L20 AND ?IMMUN?(W)?SYSTEM?(W)?STIM?
L24            94 SEA ABB=ON  L20 OR L23 AND ?HEMATOP?(W)?STEM?(W)?CELL
L25            79 SEA ABB=ON  L24 AND (?AUTOLOG? OR ?BONE?(W)?MARROW? OR ?BLOOD?
                OR ?TISSUE? OR ?ORGAN? OR ?LIVER? OR ?KIDNEY? OR ?HEART? OR
                ?CORNEA? OR ?SKIN? OR ?LUNG? OR ?PANCREAT? OR ?CULTUR?(W)?CELL?
                 OR ?SEMEN? OR EGG?)
L26            79 SEA ABB=ON  L24 AND (?BONE?(W)?MARROW? OR ?BLOOD? OR ?TISSUE?
                OR ?ORGAN? OR ?LIVER? OR ?KIDNEY? OR ?HEART? OR ?CORNEA? OR
                ?SKIN? OR ?LUNG? OR ?PANCREAT? OR ?CULTUR?(W)?CELL? OR ?SEMEN?
                OR EGG?)
L27             7 SEA ABB=ON  L26 AND ?AUTOLOG?
L28             0 SEA ABB=ON  L26 AND (?REMOV? OR ?EXTRACT? OR ?DELETE?)(3A)(?REO
                VIR?)
L29             6 SEA ABB=ON  L26 AND (?FREEZ? OR ?STOR?)
L30             1 SEA ABB=ON  L29 AND (L1 OR DMSO)
L31            79 SEA ABB=ON  L26 OR L27 OR L29 OR L30
L32             6 SEA ABB=ON  L31 AND (?METHOD? OR ?TECH? OR ?PROCED?)(3A)(?PREP?
                 OR ?DEVEL? OR ?SYNTH?)
L33            16 SEA ABB=ON  L27 OR L29 OR L30 OR L32   #6 cits from CA Plus

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO' ENTERED AT
     14:20:41 ON 03 SEP 2004
L34          6487 SEA ABB=ON  CELL?(W) COMP? AND TRANSPLANT?
L35           718 SEA ABB=ON  L34 AND (REOVIRUS? OR REOVIRID? OR VIRUS?)
L36             1 SEA ABB=ON  L35 AND ONCOLYS?(3A) RAS?
L37            11 SEA ABB=ON  L35 AND RAS?
L38             1 SEA ABB=ON  L35 AND ONCOLYS?
L39            39 SEA ABB=ON  L35 AND AUTOLOG?
L40           817 SEA ABB=ON  L14 AND (MAMMAL? OR ANIMAL? OR BIRD? OR AVIAN? OR
                HUMAN? OR SEROTYP?(W) 3 OR DEARING?(W) STRAIN?)
L41           690 DUP REMOV L40 (127 DUPLICATES REMOVED)
L42             1 SEA ABB=ON  L41 AND (ANTI?(W) REOVIRUS? OR ANTIREOVIRUS?)
L43             1 SEA ABB=ON  L41 AND IMMUN?(W) SYSTEM?(W) STIM?
L44            65 SEA ABB=ON  L41 AND HEMATOP?(W) STEM?(W) CELL?
L45           105 SEA ABB=ON  L36 OR L37 OR L38 OR L39 OR L42 OR L43 OR L44
L46            96 SEA ABB=ON  L45 AND (BONE?(W) MARROW? OR BLOOD? OR TISSUE? OR
                ORGAN? OR LIVER? OR KIDNEY? OR HEART? OR CORNEA? OR SKIN? OR
                LUNG? OR PANCREAT? OR CULTUR?(W) CELL? OR SEMEN? OR EGG?)
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L47          0 SEA ABB=ON  L46 AND (REMOV? OR EXTRACT? OR DELET?)(3A) REOVIR?
L48          5 SEA ABB=ON  L46 AND (FREEZ? OR STOR?)
L49          1 SEA ABB=ON  L48 AND (L1 OR DMSO)
L50         96 SEA ABB=ON  L46 OR L48 OR L49
L51          6 SEA ABB=ON  L50 AND (METHOD? OR TECHNIQ? OR PROCED?)(3A)(PREP?
                OR DEVEL? OR SYNTH?)
L52        *96 SEA ABB=ON  L50 OR L51

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO' ENTERED AT
     15:50:50 ON 03 SEP 2004
               SAV L52 HAR356L52/A
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6 cits from other db's

* I saved these, should you want to see additional records.

=> d ibib abs ind l13 1-4

L13 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:913021 HCAPLUS
DOCUMENT NUMBER: 139:377326
TITLE: Sensitization of neoplastic cells to radiation therapy with oncolytic viruses
INVENTOR(S): **Morris, Donald; Coffey, Matthew C.** ; **Thompson, Bradley G.**; Ball, Douglas
PATENT ASSIGNEE(S): Oncolytics Biotech Inc., Can.
SOURCE: PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003094939	A1	20031120	WO 2003-CA695	20030508
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004091463	A1	20040513	US 2003-431579	20030508
PRIORITY APPLN. INFO.:			US 2002-378948P	P 20020510
			US 2003-443189P	P 20030129

AB The present invention relates to methods of sensitizing neoplastic cells to irradiation by using oncolytic viruses, particularly reoviruses. Also provided are methods of treating or ameliorating a tumor with a combination of oncolytic viruses and radiotherapy. An example is provided of an effective treatment of nasopharyngeal cancer with radiotherapy and injection of Dearing strain reovirus at the lesion site.

IC ICM A61K035-76
ICS A61K041-00; A61P035-00
CC 8-9 (Radiation Biochemistry)
Section cross-reference(s): 1
ST reovirus sensitization tumor radiotherapy
IT Pharynx, neoplasm
(nasopharynx, carcinoma; sensitization of neoplastic cells to radiation therapy with oncolytic viruses)
IT Antitumor agents
Human
Radiosensitizers, biological
Radiotherapy
Reoviridae
(sensitization of neoplastic cells to radiation therapy with oncolytic viruses)
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:913020 HCAPLUS

DOCUMENT NUMBER: 139:375000
TITLE: Method for reducing pain using oncolytic viruses
INVENTOR(S): **Morris, Donald; Coffey, Matthew C. ; Thompson, Bradley G.**
PATENT ASSIGNEE(S): Oncolytics Biotech Inc., Can.
SOURCE: PCT Int. Appl., 40 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2003094938	A1	20031120	WO 2003-CA674		20030507
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
US 2004091458	A1	20040513	US 2003-431580		20030508
PRIORITY APPLN. INFO.:			US 2002-378675P	P	20020509
			US 2003-443177P	P	20030129

AB The invention provides a method for reducing pain associated with neoplasms in a mammal, comprising administering an effective amount of one or more oncolytic viruses. Preferably, the mammal also receives an analgesic, and the amount of analgesic required by the mammal is reduced when the oncolytic virus is administered. The oncolytic virus is preferably reovirus. The mammal may be addnl. subject to chemotherapy, immunotherapy, hormonal and/or radiation therapy. For example, a patient suffering from malignant melanoma and being permanently on narcotics received three intratumoral injections of 109 pfu of the Dearing strain of reovirus serotype 3. One week following injection, the patient reported diminished pain at the treatment site and was taken off narcotics. There was no pain at the treatment site during a 8-10 wk period after injection and no significant side effects.

IC ICM A61K035-76
ICS A61P025-04; A61P029-00; A61P035-00; A61K031-00
CC 1-6 (Pharmacology)
Section cross-reference(s): 63
ST oncolytic virus analgesic neoplasm pain
IT Bone, neoplasm
(Ewing's sarcoma; oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
IT Antitumor agents
Immunotherapy
Radiotherapy
(combination with; oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
IT Hormones, animal, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(hormonal therapy, combination with; oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)

IT Drug delivery systems
(injections; oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
IT Neoplasm
(metastasis; oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
IT Analgesics
Avian reovirus
Human
Melanoma
Pain
Reoviridae
Reovirus 1
Reovirus 2
Reovirus 3
(oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
IT Opioids
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
IT Neoplasm
(solid; oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
IT Drug interactions
(synergistic; oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
IT 57-27-2, Morphine, biological studies 57-42-1, Meperidine 76-41-5, Oxymorphone 76-42-6, Oxycodone 76-57-3, Codeine 76-99-3, Methadone 77-07-6, Levorphanol 359-83-1, Pentazocine 437-38-7, Fentanyl 466-99-9, Hydromorphone 469-62-5, Propoxyphene 20594-83-6, Nalbuphine 42408-82-2, Butorphanol 52485-79-7, Buprenorphine 53648-55-8, Dezocine
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oncolytic viruses alone or in combination with analgesics for treatment of pain associated with neoplasms)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:816871 HCAPLUS
DOCUMENT NUMBER: 135:339238
TITLE: Virus clearance of neoplastic cells from mixed cellular compositions
INVENTOR(S): **Morris, Donald; Thompson, Bradley G. ; Coffey, Matthew C.**
PATENT ASSIGNEE(S): Oncolytics Biotech, Inc., Can.
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001083710	A2	20011108	WO 2001-CA609	20010501
WO 2001083710	A3	20020502		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,

LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1278823	A2	20030129	EP 2001-931242	20010501
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2001010481	A	20030408	BR 2001-10481	20010501
JP 2003531605	T2	20031028	JP 2001-580319	20010501
US 2001048919	A1	20011206	US 2001-847355	20010503
ZA 2002008732	A	20031029	ZA 2002-8732	20021029
ZA 2002008733	A	20031029	ZA 2002-8733	20021029

PRIORITY APPLN. INFO.:

US 2000-201990P	P	20000503
US 2000-205389P	P	20000519
US 2001-268054P	P	20010213
US 2001-276782P	P	20010316
WO 2001-CA609	W	20010501

AB The present invention relates to a method for removing neoplastic cells from a mixed cellular composition, which is outside of a living organism, by using a virus which selectively infect and kill neoplastic cell. A variety of viruses can be used in this method to remove neoplastic cells for different purposes, for example, to purge hematopoietic stem cells prior to transplantation. Also provided are compns. prepared according to this method, and kits comprising a combination of viruses which are useful in this invention.

IC ICM C12N005-06
ICS C12N005-08; A01N001-02; A61L002-00; A61K035-12; C12N007-00
CC 1-6 (Pharmacology)
Section cross-reference(s): 10, 14
ST virus clearance neoplasm cell compn

IT Virus
(Delta24; virus clearance of neoplastic cells from mixed cellular compns.)

IT Gene, microbial
RL: BSU (Biological study, unclassified); PEP (Physical, engineering or chemical process); BIOL (Biological study); PROC (Process)
(E1A; virus clearance of neoplastic cells from mixed cellular compns.)

IT Virus
(Interferon sensitive; virus clearance of neoplastic cells from mixed cellular compns.)

IT Virus
(ONYX-015; virus clearance of neoplastic cells from mixed cellular compns.)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Rb; virus clearance of neoplastic cells from mixed cellular compns.)

IT Virus
(Replication competent; virus clearance of neoplastic cells from mixed cellular compns.)

IT Eye
(cornea; virus clearance of neoplastic cells from mixed cellular compns.)

IT Gene
(expression; virus clearance of neoplastic cells from mixed cellular compns.)

IT Mammary gland
(neoplasm; virus clearance of neoplastic cells from mixed cellular compns.)

IT Parapoxvirus
(orf; virus clearance of neoplastic cells from mixed cellular compns.)
IT Hematopoietic precursor cell
(stem; virus clearance of neoplastic cells from mixed cellular compns.)
IT Adenoviridae
Animal cell
Animal tissue
Animal tissue culture
Apoptosis
Blood
Bone marrow
Cell differentiation
Cell proliferation
Composition
Egg
Heart
Human herpesvirus
Infection
Kidney
Liver
Lung
Mutation
Neoplasm
Newcastle disease virus
Organ, animal
Pancreatic islet of Langerhans
Reoviridae
Semen
Skin
Solutions
Storage
Test kits
Translation, genetic
Transplant and Transplantation
Vaccinia virus
Vesicular stomatitis virus
Virus
(virus clearance of neoplastic cells from mixed cellular compns.)
IT CD34 (antigen)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(virus clearance of neoplastic cells from mixed cellular compns.)
IT Proteins, general, biological studies
p53 (protein)
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(virus clearance of neoplastic cells from mixed cellular compns.)
IT Interferons
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(virus clearance of neoplastic cells from mixed cellular compns.)
IT 37211-65-7, RNA kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(double stranded; virus clearance of neoplastic cells from mixed cellular compns.)
IT 67-68-5, DMSO, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(virus clearance of neoplastic cells from mixed cellular compns.)

L13 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2001:813370 HCAPLUS
TITLE: Reovirus clearance of ras-mediated neoplastic cells from mixed cellular compositions
INVENTOR(S): **Morris, Donald; Thompson, Bradley G.; Coffey, Matthew C.**
PATENT ASSIGNEE(S): Oncolytics Biotech, Inc., Can.
SOURCE: PCT Int. Appl.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001083711	A2	20011108	WO 2001-CA620	20010502
WO 2001083711	A3	20020510		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1278824	A2	20030129	EP 2001-931251	20010502
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2001010474	A	20030401	BR 2001-10474	20010502
JP 2003531606	T2	20031028	JP 2001-580320	20010502
US 2002006398	A1	20020117	US 2001-847356	20010503
ZA 2002008732	A	20031029	ZA 2002-8732	20021029
ZA 2002008733	A	20031029	ZA 2002-8733	20021029

PRIORITY APPLN. INFO.:

US 2000-201990P	P	20000503
US 2000-205389P	P	20000519
US 2001-268054P	P	20010213
WO 2001-CA620	W	20010502

AB Reovirus can be used to selectively remove ras-mediated neoplastic cells from a cellular composition. It is of particular interest to purge autographs which may contain neoplastic cells with reovirus before transplanting the autografts back into the recipient, thereby reducing the risk of introducing or reintroducing neoplastic cells into the recipient.

IC ICM C12N005-06
ICS C12N005-08; A01N001-02; A61L002-00; A61K035-12; A61K039-42; A61K039-42; A61K035-12

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=> d que stat l33
L1             1 SEA FILE=REGISTRY ABB=ON  "LINOLEIC ACID"/CN
L13          894 SEA FILE=HCAPLUS ABB=ON  ?CELL?(W)?COMP? AND ?TRANSPLANT?
L14          105 SEA FILE=HCAPLUS ABB=ON  L13 AND (?REOVIRUS? OR ?REOVIRIDAE?
                 OR ?VIRUS?)
L16            7 SEA FILE=HCAPLUS ABB=ON  L14 AND RAS?
L18            7 SEA FILE=HCAPLUS ABB=ON  L14 AND ?AUTOLOG?
L19           92 SEA FILE=HCAPLUS ABB=ON  L14 AND (?MAMMAL? OR ?ANIMAL? OR
                 ?AVIAN? OR ?BIRD? OR ?HUMAN? OR ?SEROTYP?(W)3 OR ?DEARING?(W)?S
                 TRAIN?)
L20           94 SEA FILE=HCAPLUS ABB=ON  L16 OR L18 OR L19
L23            1 SEA FILE=HCAPLUS ABB=ON  L20 AND ?IMMUN?(W)?SYSTEM?(W)?STIM?
L24           94 SEA FILE=HCAPLUS ABB=ON  L20 OR L23 AND ?HEMATOP?(W)?STEM?(W)?C
                 ELL
L26           79 SEA FILE=HCAPLUS ABB=ON  L24 AND (?BONE?(W)?MARROW? OR ?BLOOD?
                 OR ?TISSUE? OR ?ORGAN? OR ?LIVER? OR ?KIDNEY? OR ?HEART? OR
                 ?CORNEA? OR ?SKIN? OR ?LUNG? OR ?PANCREAT? OR ?CULTUR?(W)?CELL?
                  OR ?SEMEN? OR EGG?)
L27            7 SEA FILE=HCAPLUS ABB=ON  L26 AND ?AUTOLOG?
L29            6 SEA FILE=HCAPLUS ABB=ON  L26 AND (?FREEZ? OR ?STOR?)
L30            1 SEA FILE=HCAPLUS ABB=ON  L29 AND (L1 OR DMSO)
L31           79 SEA FILE=HCAPLUS ABB=ON  L26 OR L27 OR L29 OR L30
L32            6 SEA FILE=HCAPLUS ABB=ON  L31 AND (?METHOD? OR ?TECH? OR
                 ?PROCED?)(3A)(?PREP? OR ?DEVEL? OR ?SYNTH?)
L33           16 SEA FILE=HCAPLUS ABB=ON  L27 OR L29 OR L30 OR L32

=> d ibib abs l33 1-16

L33  ANSWER 1 OF 16  HCAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:        2004:571323  HCAPLUS
TITLE:                   Enhanced cytotoxicity of allogeneic NK cells with
                         killer immunoglobulin-like receptor ligand
                         incompatibility against melanoma and renal cell
                         carcinoma cells
AUTHOR(S):               Igarashi, Takehito; Wynberg, Jason; Srinivasan,
                         Ramprasad; Becknell, Brian; McCoy, J. Phillip, Jr.;
                         Takahashi, Yoshiyuki; Suffredini, Dante A.; Linehan,
                         W. Marston; Caligiuri, Michael A.; Childs, Richard W.
CORPORATE SOURCE:        Hematology Branch, Flow Cytometry Core Facility,
                         National Heart, Lung and Blood Institute, National
                         Institutes of Health, Bethesda, MD, USA
SOURCE:                  Blood (2004), 104(1), 170-177
                         CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER:               American Society of Hematology
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   Cellular inactivation through killer Ig-like receptors (KIRs) may allow
     neoplastic cells to evade host natural killer (NK) cell-mediated immunity.
     Recently, alloreactive NK cells were shown to mediate antileukemic effects
     against acute myelogenous leukemia (AML) after mismatched
     transplantation, when KIR ligand incompatibility existed in the
     direction of graft-vs.-host disease (GVHD). Therefore, we investigated
     whether solid tumor cells would have similar enhanced susceptibility to
     allogeneic KIR-incompatible NK cells compared with
     their KIR-matched autologous or allogeneic counterparts. NK
     populations enriched and cloned from the blood of cancer
     patients or healthy donors homozygous for HLA-C alleles in group 1 (C-G1)
     or group 2 (C-G2) were tested in vitro for cytotoxicity against
     Epstein-Barr virus-transformed lymphoblastic cell lines
     (EBV-LCLs), renal cell carcinoma (RCC), and melanoma (MEL) cells with or
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without a matching KIR-inhibitory HLA-C ligand. Allogeneic NK cells were more cytotoxic to tumor targets mismatched for KIR ligands than their KIR ligand-matched counterparts. Bulk NK populations (CD3-/CD2+/CD56+) expanded 104-fold from patients homozygous for C-G1 or C-G2 had enhanced cytotoxicity against KIR ligand-mismatched tumor cells but only minimal cytotoxicity against KIR ligand-matched targets. Further, NK cell lines from C-G1 or C-G2 homozygous cancer patients or healthy donors expanded but failed to kill **autologous** or KIR-matched MEL and RCC cells yet had significant cytotoxicity (more than 50% lysis at 20:1 effector-target [E/T] ratio) against allogeneic KIR-mismatched tumor lines. These data suggest immunotherapeutic strategies that use KIR-incompatible allogeneic NK cells might have superior antineoplastic effects against solid tumors compared with approaches using **autologous** NK cells.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 2 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:96721 HCAPLUS
DOCUMENT NUMBER: 139:219036
TITLE: Biologic **liver** support: optimal cell source and mass
AUTHOR(S): Morsiani, E.; Brogli, M.; Galavotti, D.; Pazzi, P.; Puviani, A. C.; Azzena, G. F.
CORPORATE SOURCE: Department of Surgery, Sant'Anna University Hospital, Ferrara, Italy
SOURCE: International Journal of Artificial Organs (2002), 25(10), 985-993
CODEN: IJAODS; ISSN: 0391-3988
PUBLISHER: Wichtig Editore
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Hepatic support is indicated in acute **liver** failure (ALF) patients to foster **liver** regeneration, or until a **liver** becomes available for orthotopic-**liver transplantation** (OLT), in primary non function of the **transplanted liver**, and hopefully in chronic **liver** disease patients affected by ALF episodes, in whom OLT is not a therapeutic option. The concept of bioartificial **liver** (BAL) is based on the assumption that only the hepatocytes can perform the whole spectrum of biotransformation functions, which are needed to prevent hepatic encephalopathy, coma and cerebral edema. Among others, two important issues are related to BAL development: i) the choice of the **cellular component**; 2) the cell mass needed to perform an adequate BAL treatment. Primary hepatocytes, of **human** or **animal** origin, should be considered the first choice because they express highly differentiated functions. Accordingly, a minimal cell mass corresponding to 10% of a **human** adult **liver**, i.e. 150 g of freshly isolated, ≥90% viable hepatocytes should be used. When 4 °C cold-**stored** or cryopreserved hepatocytes are used, the cellular mass should be increased because of a drop in cell viability and function. In case of hepatoma-derived cells, **cultured cell** lines or engineered cells, an adequate functional cell mass should be used, expressing metabolic and biotransformation activities comparable to those of primary hepatocytes. Finally, the use of porcine hepatocytes or other **animal** cells in BAL devices should be presently directed only to ALF patients as a bridge treatment to OLT, because of potential transmission of **animal retrovirus** and prions which may potentially cause major pandemics.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 3 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:787928 HCAPLUS
DOCUMENT NUMBER: 138:105549
TITLE: Isolation and expansion of **human cytomegalovirus**-specific cytotoxic T lymphocytes using interferon-γ secretion assay
AUTHOR(S): Bissinger, Alfred Lennart; Rauser, Georg; Hebart, Holger; Frank, Friederike; Jahn, Gerhard; Einsele, Hermann
CORPORATE SOURCE: Medizinische Klinik II, Eberhard-Karls-Universitat Tuebingen, Tuebingen, D-72076, Germany
SOURCE: Experimental Hematology (New York, NY, United States) (2002), 30(10), 1178-1184
CODEN: EXHMA6; ISSN: 0301-472X
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The aim of this study was to isolate and expand donor-derived **human cytomegalovirus** (HCMV)-specific cytotoxic T lymphocytes (CTLs) for adoptive transfer of 107 cells per m2 of body surface area to **restore** protective immunity after stem cell **transplantation.** A new strategy to generate HCMV-specific CTLs using the interferon-γ (IFN-γ) secretion assay, followed by expansion to nos. sufficient for clin. application with interleukin-2 and feeder cell stimulation, is described. From 1 to 5 + 104 HCMV peptide-specific T lymphocytes (greater than 90% CD3+CD8+) were isolated from 1 to 2 + 108 peripheral **blood** mononuclear **cells comparable** to 50 to 100 mL of **blood** from HLA-A*0201 HCMV seropos. **blood** donors (n= 14) and expanded ex vivo after a median of 16 days (range 8-28 days; n= 13) to greater than 107/m2 HCMV peptide-specific CTLs using **autologous** (n= 2) or allogeneic (n = 11) feeder cell stimulation. In three expts., expansion to 6 wk was performed, achieving a median of 1.6 + 109 cells (range 6.1 + 108-3.3 + 109). Characterization of these HCMV-specific CTL lines revealed an average purity of 89.2% (range 66.2-99.3%) using HCMV pp65 peptide HLA-A*0201 tetramer staining (n= 14) and 89.4% (range 64.4-99.5%) by peptide-specific IFN-γ secretion (n= 7). A median of 82.6% (range 76.0-88.0%) showed perforin secretion (n = 3) and 57.5% (range 22.2-80.7%) specific lysis of peptide-pulsed T2 cells (n = 5). A median of 52.2% (range 35.2-7.3%) revealed specific killing of HCMV-infected **autologous**, but not allogeneic, fibroblasts (n = 6). IFN-γ secretion assay allows development of a simple and rapid protocol with short expansion times for generation of greater than 107/m2 HCMV-specific CTLs for adoptive immunotherapy.
REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 4 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:716032 HCAPLUS
DOCUMENT NUMBER: 137:231752
TITLE: Compositions and methods for modifying the content of polyunsaturated fatty acids in **mammalian** cells
INVENTOR(S): Kang, Jing X.
PATENT ASSIGNEE(S): The General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 60 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2002072028	A2	20020919	WO 2002-US7649		20020312
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
US 2004115681	A1	20040617	US 2004-468318		20040112
PRIORITY APPLN. INFO.:			US 2001-275222P	P	20010312
			WO 2002-US7649	W	20020312

AB The present invention features compns. (e.g., nucleic acids encoding fat-1, optionally and operably linked to a constitutively active or **tissue**-specific promoter or other regulatory sequence and pharmaceutically acceptable formulations including that nucleic acid or biol. active variants thereof) and methods that can be used to effectively modify the content of PUFAs in **animal** cells (i.e., cells other than those of C. elegants, for example, **mammalian** cells such as myocytes, neurons (whether of the peripheral or central nervous system), adipocytes, endothelial cells, and cancer cells). The modified cells, whether in vivo or ex vivo (e.g., in **tissue** culture), transgenic **animals** containing them, and food products obtained from those **animals** (e.g., meat or other edible parts of the **animals** (e.g., **liver**, **kidney**, or sweetbreads)) are also within the scope of the present invention.

L33 ANSWER 5 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:196122 HCAPLUS
DOCUMENT NUMBER: 136:308248
TITLE: Comparison of five **retrovirus** vectors containing the **human** IL-2 receptor γ chain gene for their ability to **restore** T and B lymphocytes in the X-linked severe combined immunodeficiency mouse model
AUTHOR(S): Mendoza, Guillermo J. Aviles; Seidel, Nancy E.; Otsu, Makoto; Anderson, Stacie M.; Simon-Stoos, Karen; Herrera, Adrianna; Hoogstraten-Miller, Shelley; Malech, Harry L.; Candotti, Fabio; Puck, Jennifer M.; Bodine, David M.
CORPORATE SOURCE: Hematopoiesis Section, Genetics and Molecular Biology Branch, National Human Genome Research Institute, National Institutes of Health, Bethesda, MD, 20892, USA
SOURCE: Molecular Therapy (2001), 3(4), 565-573
CODEN: MTOHCK; ISSN: 1525-0016
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB X-linked severe combined immunodeficiency (XSCID) is caused by mutations in the IL-2 receptor γ chain (IL2RG) gene, resulting in absent T lymphocytes and nonfunctional B lymphocytes. Recently T lymphocyte production and B lymphocyte function were **restored** in XSCID patients

infused with **autologous** stem cells transduced with a **retrovirus** containing the **human** IL2RG cDNA. To optimize the expression of **human** IL2RG for future clin. trials, we compared five retroviral vectors expressing **human** IL2RG from different LTR enhancer-promoter elements in a mouse model. Northern and Southern blot anal. of hematopoietic **tissues** from repopulated mice revealed that the retroviral vector with the highest expression per copy number was MFG-S-hIL2RG, followed by MND-hIL2RG. All five vectors were capable of **restoring** lymphopoiesis in irradiated XSCID mice **transplanted** with transduced IL2RG-deficient hematopoietic stem cells. Transduction of IL2RG-deficient hematopoietic stem cells with all five vectors **restored** T lymphopoiesis in **transplanted** stem cell-deficient W/Wv mouse recipients. However, only XSCID stem cells transduced with the MFG-S-hIL2RG vector generated B lymphocytes in W/Wv mice. We conclude that the MFG-S-hIL2RG vector provides the best opportunity for in vivo selection and development of B and T lymphocytes for **human** XSCID gene therapy. (c) 2001 Academic Press.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 6 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:185608 HCAPLUS
DOCUMENT NUMBER: 136:242941
TITLE: DNA transfer from apoptotic bodies of donor cells to engulfing recipient cells
INVENTOR(S): Spetz-Holmgren, Anna-Lena; Holmgren, Lars; Andersson, Jan; Folkman, Judah
PATENT ASSIGNEE(S): Swed.
SOURCE: U.S. Pat. Appl. Publ., 47 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2002031521	A1	20020314	US 2001-842073		20010426
US 6506596	B2	20030114			
PRIORITY APPLN. INFO.:			US 2000-208326P	P	20000601

AB The present invention relates to a method of of transferring genomic DNA from apoptotic bodies to engulfing cells, wherein DNA is transferred from a donor cell to a recipient cell. More specifically the method includes providing somatic donor **cells comprising** desired DNA; generating apoptotic bodies of said donor cells; incubation of the apoptotic bodies with engulfing recipient cells under biol. conditions allowing uptake of DNA from the apoptotic bodies by said recipient cells; and optionally selecting recipient cells which have integrated DNA from the apoptotic bodies. The present method is useful in various pharmaceutical applications, such as in vaccine **prepns.** and gene identification **procedures.** Further, the present invention also relates to a method of preventing and/or treating a clin. condition in a patient, which comprises administering the recipient cells in a pharmaceutically acceptable carrier to the patient, thus enabling a protective and/or therapeutic reaction against the clin. condition.

L33 ANSWER 7 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:816871 HCAPLUS
DOCUMENT NUMBER: 135:339238
TITLE: **Virus** clearance of neoplastic cells from

mixed **cellular compositions**
INVENTOR(S): Morris, Donald; Thompson, Bradley G.; Coffey, Matthew C.
PATENT ASSIGNEE(S): Oncolytics Biotech, Inc., Can.
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001083710	A2	20011108	WO 2001-CA609	20010501
WO 2001083710	A3	20020502		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1278823	A2	20030129	EP 2001-931242	20010501
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2001010481	A	20030408	BR 2001-10481	20010501
JP 2003531605	T2	20031028	JP 2001-580319	20010501
US 2001048919	A1	20011206	US 2001-847355	20010503
ZA 2002008732	A	20031029	ZA 2002-8732	20021029
ZA 2002008733	A	20031029	ZA 2002-8733	20021029

PRIORITY APPLN. INFO.:

US 2000-201990P	P	20000503
US 2000-205389P	P	20000519
US 2001-268054P	P	20010213
US 2001-276782P	P	20010316
WO 2001-CA609	W	20010501

AB The present invention relates to a method for removing neoplastic cells from a mixed **cellular composition**, which is outside of a living **organism**, by using a **virus** which selectively infect and kill neoplastic cell. A variety of **viruses** can be used in this method to remove neoplastic cells for different purposes, for example, to purge hematopoietic stem cells prior to **transplantation.** Also provided are compns. **prepared** according to this **method**, and kits comprising a combination of **viruses** which are useful in this invention.

L33 ANSWER 8 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:536372 HCAPLUS
DOCUMENT NUMBER: 136:165615
TITLE: **Cytomegalovirus** infectivity in whole **blood** following leukocyte reduction by filtration
AUTHOR(S): Lipson, Steven M.; Shepp, David H.; Match, Mark E.; Axelrod, Frederick B.; Whitbread, John A.
CORPORATE SOURCE: Departments of Laboratories, North Shore University Hospital-NYU School of Medicine, Manhasset, NY, 11234, USA
SOURCE: American Journal of Clinical Pathology (2001), 116(1), 52-55

CODEN: AJCPAI; ISSN: 0002-9173
PUBLISHER: American Society of Clinical Pathologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Cytomegalovirus** (CMV) may be transmitted by transfusion of whole **blood** and **cellular components** processed according to standard processing procedures. A need exists to **develop** new **procedures** to remove CMV and other leukocyte-borne **viruses** from donor **blood.** Ten patients (AIDS/ **bone marrow transplants**) who were CMV antigenemic (**virus** subsequently confirmed by isolation), donated 50 mL of venous **blood** within 24 to 72 h of the initial antigen detection. Twenty-five-milliliter aliquots of each specimen were passed through Purecell Neo Neonatal Leukocyte Reduction Filters (Pall, East Hills, NY). The remaining 25-mL nonfiltered aliquots, as well as the **blood** filtrates, were subjected to infectivity endpoint detns. The Purecell Neo filter effected a 3 to 4 log10 leukocyte reduction CMV input titers ranged from less than 10 to 7.3 + 101 median **tissue** culture infectious dose (TCID50) per mL. CMV was not isolated from any postfiltration effluent (ie, leukocytes, erythrocytes, or plasma). CMV DNA was not detected by nested polymerase chain reaction in 8 of 10 postfiltrate **blood** specimens. The Purecell Neo filter was efficacious in eliminating or significantly reducing viral (CMV) load in venous **blood.**
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 9 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:212638 HCAPLUS
DOCUMENT NUMBER: 134:352112
TITLE: Interleukin-7 **restores** immunity in athymic T-cell-depleted hosts
AUTHOR(S): Fry, Terry J.; Christensen, Barbara L.; Komschlies, Kristin L.; Gress, Ronald E.; Mackall, Crystal L.
CORPORATE SOURCE: Molecular Oncology Section, Pediatric Branch, National Cancer Institute, National Institutes of Heath, Bethesda, MD, USA
SOURCE: Blood (2001), 97(6), 1525-1533
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Thymic-deficient hosts rely primarily on antigen-driven expansion to **restore** the peripheral T-**cell compartment** following T-cell depletion (TCD). The degree to which this thymic-independent pathway can **restore** immune competence remains poorly understood but has important implications for a number of clin. conditions including stem cell **transplantation** and **human** immunodeficiency **virus** (HIV) infection. A model of HY-mediated **skin** graft rejection by athymic, TCD mice was used to show that **restoration** of naive and recall responses via peripheral expansion requires transfer of only 25 + 106 lymph node (LN) cells representing approx. 10% of the T-cell repertoire. Constitutive expression of bcl-2 in the expanding inocula **restored** recall responses to HY at a substantially lower LN cell dose (1 + 106), which is normally insufficient to induce HY-mediated graft rejection in athymic hosts. Interestingly, bcl-2 had no effect on primary responses. Interleukin-7 (IL-7) potently enhanced thymic-independent peripheral expansion and led to HY graft rejection using an LN cell dose of 1 + 106 in both primary and recall models. The **restoration** of

immune competence by IL-7 appeared to be mediated through a combination of programmed cell death inhibition, improved costimulation, and modulation of antigen-presenting cell (APC) function. These results show that immune competence for even stringent antigens such as HY can be **restored** in the absence of thymic function and identify IL-7 as a potent modulator of thymic-independent T-cell regeneration.

REFERENCE COUNT: 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 10 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:61825 HCAPLUS
DOCUMENT NUMBER: 132:217699
TITLE: Marking and gene expression by a **lentivirus** vector in **transplanted human** and **nonhuman** primate CD34+ cells
AUTHOR(S): An, Dong Sung; Wersto, Robert P.; Agricola, Brian A.; Metzger, Mark E.; Lu, Stephanie; Amado, Rafael G.; Chen, Irvin S. Y.; Donahue, Robert E.
CORPORATE SOURCE: ULCA AIDS Institute, University of California, Los Angeles, Los Angeles, CA, USA
SOURCE: Journal of Virology (2000), 74(3), 1286-1295
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Recently, gene **delivery** vectors based on **human** immunodeficiency **virus** (HIV) have been developed as an alternative mode of gene **delivery.** These vectors have a number of advantages, particularly in regard to the ability to infect cells which are not actively dividing. However, the use of vectors based on **human** immunodeficiency **virus** raises a number of issues, not the least of which is safety; therefore, further characterization of marking and gene expression in different hematopoietic lineages in primate **animal** model systems is desirable. We use two **animal** model systems for gene therapy to test the efficiency of transduction and marking, as well as the safety of these vectors. The first utilizes the rhesus **animal** model for cytokine-mobilized **autologous** peripheral **blood** CD34+ cell **transplantation.** The second uses the SCID-**human** (SCID-hu) thymus/**liver** chimeric graft **animal** model useful specifically for **human** T-lymphoid progenitor cell reconstitution. In the rhesus macaques, detectable levels of vector were observed in granulocytes, lymphocytes, monocytes, and, in one **animal** with the highest levels of marking, erythrocytes and platelets. In **transplanted** SCID-hu mice, we directly compared marking and gene expression of the **lentivirus** vector and a murine leukemia **virus**-derived vector in thymocytes. Marking was observed at comparable levels, but the **lentivirus** vector bearing an internal **cytomegalovirus** promoter expressed less efficiently than did the murine retroviral vector expressed from its own long terminal repeats. In assays for infectious HIV type 1 (HIV-1), no replication-competent HIV-1 was detected in either **animal** model system. Thus, these results indicate that while **lentivirus** vectors have no apparent deleterious effects and may have advantages over murine retroviral vectors, further study of the requirements for optimal use are warranted.

REFERENCE COUNT: 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 11 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:37382 HCAPLUS

DOCUMENT NUMBER: 130:222080
TITLE: Comparison of immune reconstitution after unrelated and related T-cell-depleted **bone marrow transplantation**: effect of patient age and donor leukocyte infusions
AUTHOR(S): Small, T. N.; Papadopoulos, E. B.; Boulad, F.; Black, P.; Castro-Malaspina, H.; Childs, B. H.; Collins, N.; Gillio, A.; George, D.; Jakubowski, A.; Heller, G.; Fazzari, M.; Kernan, N.; MacKinnon, S.; Szabolcs, P.; Young, J. W.; O'Reilly, R. J.
CORPORATE SOURCE: Memorial Sloan-Kettering Cancer Center, New York, NY, 10021, USA
SOURCE: Blood (1999), 93(2), 467-480
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Unrelated **bone marrow transplantation** (BMT) is often complicated by fatal opportunistic infections. To evaluate features unique to immune reconstitution after unrelated BMT, the lymphoid phenotype, in vitro function, and life-threatening opportunistic infections after unrelated and related T-cell-depleted (TCD) BMT were analyzed longitudinally and compared. The effects of **posttransplant** donor leukocyte infusions to treat or prevent **cytomegalovirus** (CMV) or Epstein-Barr **virus** (EBV) infections on immune reconstitution were also analyzed. This study demonstrates that adult recipients of TCD unrelated BMTs experience prolonged and profound deficiencies of CD3+, CD4+, and CD8+ T-cell populations when compared with pediatric recipients of unrelated BMT and adults after related BMT ($P < .01$), that these adults have a significantly increased risk of life-threatening opportunistic infections, and that the rate of recovery of CD4 T cells correlates with the risk of developing these infections. Recovery of normal nos. of CD3+, CD8+, and CD4+ T-cell populations is similar in children after related or unrelated BMT. This study also demonstrates that adoptive immunotherapy with small nos. of unirradiated donor leukocytes can be associated with rapid **restoration** of CD3+, CD4+, and CD8+ T-cell nos., antigen-specific T-cell responses, and resolution of CMV- and EBV-associated disease after unrelated TCD BMT.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 12 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:205254 HCAPLUS
DOCUMENT NUMBER: 126:198546
TITLE: **Autologous** immune cell therapy: **cell compositions**, methods and applications to treatment of **human** disease
INVENTOR(S): Gruenberg, Michael L.
PATENT ASSIGNEE(S): Celltherapy, Inc., USA; Gruenberg, Michael L.
SOURCE: PCT Int. Appl., 98 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9705239	A1	19970213	WO 1996-US12170	19960725

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM

CA 2227327	AA	19970213	CA 1996-2227327		19960724
JP 2001520509	T2	20011030	JP 1997-507706		19960724
AU 9666499	A1	19970226	AU 1996-66499		19960725
EP 852618	A1	19980715	EP 1996-926117		19960725
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI					
US 2002182730	A1	20021205	US 1998-127411		19980731
US 2001031253	A1	20011018	US 2001-824906		20010402
US 2003039650	A1	20030227	US 2002-155404		20020522
PRIORITY APPLN. INFO.:			US 1995-506668	A	19950725
			US 1995-44693P	P	19950726
			US 1996-700565	A3	19960725
			WO 1996-US12170	W	19960725
			US 1998-127138	A1	19980731

AB Compns. containing substantially homogeneous populations of functionally or phenotypically defined immune cells that have been isolated from a patient and expanded and/or differentiated ex vivo. The immune cells are effector or memory or regulatory T cells, Th1 cells, Th2 cells, Th3 cells, CD4+ cells, CD8+ cells, etc. The cell population expansion is activated by sp. surface protein, interferon-γ, interleukin 2, interleukin 4, anti-γ interferon, anti-interleukin 12, monoclonal antibody to CD3, CD2, CD4, CD8, CD11a, CD27, CD28, CD44, or CD45RO, and is performed in a hollow fiber bioreactor. Methods for treating or preventing disease or otherwise altering the immune status of the patient by reinfusing such cells into the donor are also provided. The **autologous** immune cell therapy is used for treating autoimmune disease, chronic inflammation, allergy, infection, **organ** or **tissue** **transplant** rejection, rheumatoid arthritis, inflammatory bowel disease, insulin-dependent diabetes mellitus, tumor, multiple sclerosis, Crohn's disease, HIV infection, etc.

L33 ANSWER 13 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:673855 HCAPLUS
DOCUMENT NUMBER: 121:273855
TITLE: Improved method for gene transfer into **mammalian** cells and use of transfected cells in gene therapy and **transplantation**
INVENTOR(S): Dube, Ian D.; Kamel-Reid, Suzanne
PATENT ASSIGNEE(S): Can.
SOURCE: Can. Pat. Appl., 38 pp.
CODEN: CPXXEB
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CA 2086844	AA	19940708	CA 1993-2086844	19930107
PRIORITY APPLN. INFO.:			CA 1993-2086844	19930107

AB A method of effecting transfer of a gene into **mammalian** cells, particularly hematopoietic cells, with a gene transfer vehicle, particularly a retroviral vector is described. The method comprises establishing a long term cell culture and exposing the culture to

multiple, periodic infections of the vector containing the gene and, preferably, comprising multiple, periodic partial substitutions of the medium and cells. Genetically marked cells are returned to **autologous** recipients in the absence of any type of conditioning. The method provides improved gene transfer efficiency without increased toxicity. The method was demonstrated with Moloney murine leukemia **virus**-derived vector N2 infection of canine mononuclear cells followed by **transplantation** of these transgenic cells into dogs. The results of these expts. indicated that long-term marrow culture (LTMC) cells could reconstitute the hematopoietic system of dogs; marrow ablative conditioning is not necessary for engraftment of the LTMC cells and may, in fact, compromise engraftment by upregulating endogenous hematopoiesis; only a few stem cells are cycling at any given time in dogs; and in vitro activated stem **cells complete** normal differentiation and proliferation programs when returned to the in vivo microenvironments from whence they came.

L33 ANSWER 14 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1991:654148 HCAPLUS
DOCUMENT NUMBER: 115:254148
TITLE: Methods and compositions for promoting immunopotentiation
INVENTOR(S): Bluestone, Jeffery A.
PATENT ASSIGNEE(S): Arch Development Corp., USA
SOURCE: PCT Int. Appl., 112 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9106319	A1	19910516	WO 1990-US6177	19901026
W: AT, AU, BB, BG, BR, CA, CH, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MC, MG, MW, NL, NO, RO, SD, SE, SU				
RW: AT, BE, BF, BJ, CF, CG, CH, CM, DE, DK, ES, FR, GA, GB, GR, IT, LU, ML, MR, NL, SE, SN, TD, TG				
CA 2071478	AA	19910428	CA 1990-2071478	19901026
AU 9066423	A1	19910531	AU 1990-66423	19901026
EP 497883	A1	19920812	EP 1990-916853	19901026
EP 497883	B1	19980715		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
JP 05504554	T2	19930715	JP 1990-515665	19901026
JP 2546544	B2	19961023		
EP 839536	A1	19980506	EP 1998-100138	19901026
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
AT 168272	E	19980815	AT 1990-916853	19901026
US 6113901	A	20000905	US 1994-286805	19940805
US 6143297	A	20001107	US 1995-458462	19950602
US 6406696	B1	20020618	US 1995-459486	19950602
US 2003165542	A1	20030904	US 2002-67104	20020204

PRIORITY APPLN. INFO.:

US 1989-429729	A	19891027
US 1990-524304	A	19900516
EP 1990-916853	A3	19901026
WO 1990-US6177	A	19901026
US 1992-990553	B1	19921214
US 1994-286805	A3	19940805
US 1995-459486	A3	19950602

AB This invention discloses immunopotentiating agents which stimulate an

immune response. These agents are single agents that act directly, adjuvants added concurrently with the agents, or heteroconjugates. Heteroconjugate agents elicit or enhance a cellular or humoral immune response which may be specific for an epitope contained within an amino acid sequence. Enhanced hematopoieses by **bone marrow** stem cell recruitment was also a result of administering some of these agents. Examples of immunopotentiating agents include monoclonal antibodies and proteins derived from **microorganisms** (e.g., enterotoxins) which activate T-cells. One method of treatment disclosed uses only the immunopotentiating agent to stimulate the immune system. Another uses adjuvants in combination with the agent. A third method employs heteroconjugates comprising (a) an immunopotentiating protein which is characterized as having an ability to stimulate T-cells; and (b) a second protein having an amino acid sequence which includes an epitope against which a cellular or humoral response is desired. This invention also relates to a **method** of **preparing** the heteroconjugate, and to a method of stimulating the immune system in vivo in a novel way. One route of stimulation is to activate T-cells, in some instances, specific subsets of T-cells, by administering heteroconjugates containing an immunopotentiating protein and a second protein, to **mammals.** For this method of treatment, the second protein in the heteroconjugate is derived from abnormal or diseased **tissue**, or from an infectious agent; alternatively, the second protein is produced **synthetically** by standard **methods** of mol. biol. Sources of the second protein include tumors, **viruses**, bacteria, fungi, protozoal or metozoal parasites. Monoclonal antibodies or T-cells prepared from **mammals** whose immune systems have responsed to administration of the heteroconjugate may be produced and administered to induce passive immunity. A **method** of **preparing** a hybridoma which secretes the monoclonal antibodies and use of these monoclonal antibodies and T-cells, are also disclosed. This invention is also directed to a vaccine comprising the heteroconjugate. Administration of low doses of monoclonal anti-CD3 prevented lethal pneumonia caused by Sendai **virus** in >60% of mice. Anti-CD3-treated, virally-infected mice also developed lasting **virus**-specific immunity. The 129/J strain of mice was also protected.

L33 ANSWER 15 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1991:509477 HCAPLUS
DOCUMENT NUMBER: 115:109477
TITLE: The immunoregulatory effects of merocyanine 540 on in vitro **human** T- and B-lymphocyte functions
AUTHOR(S): Lum, Lawrence G.; Yamagami, Masahiko; Giddings, Bernadette R.; Joshi, Indira; Schober, Sheri L.; Sensenbrenner, Lyle L.; Sieber, Fritz
CORPORATE SOURCE: Dep. Med., Wayne State Univ., Detroit, MI, 48202-0188, USA
SOURCE: Blood (1991), 77(12), 2701-6
CODEN: BLOOAW; ISSN: 0006-4971
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Merocyanine 540 (MC 540) is a photoactive dye used to purge **bone marrow** of tumor cells in **autologous bone marrow transplantation.** The effects of MC 540 on the lymphoid components in the marrow are unknown. This study evaluates the treatment of lymphocytes by MC 540 (15 μg/mL) and light (70 W/m2) on: (1) phytohemagglutinin and Con A-induced proliferation; (2) allogeneic mixed lymphocyte cultures (MLC); (3) the regulation of Ig synthesis by T cells; and (4) the ability of B cells to produce polyclonal Igs as measured by an ELISA-plaque assay. The results show that MC 540 and light

treatment reduced Con A-stimulated T-cell proliferation greater than 50% after 30 min and greater than 80% after 60 min of MC 540-sensitized photoirradn. Ninety minutes of MC 540 and light exposure (designated treatment) inhibited MLC greater than 90%. In polyclonal Ig synthesis, T-cell helper activity could be abrogated by 90 min of treatment in cocultures containing untreated B cells. Purified B cells treated for 90 min cultured with normal T cells did not produce Ig. Treatment of B **cells completely** inhibited Epstein-Barr **virus** -stimulated Ig synthesis. These data show that T- and B-cell immunity is suppressed by the MC 540-sensitized photoirradn. Treatment of **bone marrow** with MC 540 and light may have profound effects on immune reconstitution in **autologous** marrow graft recipients. More provocative is the fact that the same immunomodulatory effects may be applicable to partially mismatched marrow **transplant** situations as a means of reducing graft-vs.-host reactions.

L33 ANSWER 16 OF 16 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1990:584733 HCAPLUS
DOCUMENT NUMBER: 113:184733
TITLE: Luminide and macroluminide class of pharmaceuticals
INVENTOR(S): Mills, Randell L.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 274 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 8909833	A1	19891019	WO 1989-US1361	19890331
W: AU, HU, JP, SU				
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
AU 8934454	A1	19891103	AU 1989-34454	19890331
EP 414730	A1	19910306	EP 1989-904951	19890331
EP 414730	B1	19991215		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
JP 03505574	T2	19911205	JP 1989-504746	19890331
JP 3025817	B2	20000327		
AT 187776	E	20000115	AT 1989-904951	19890331
CN 1047075	A	19901121	CN 1989-103146	19890510
CN 1089086	B	20020814		
PRIORITY APPLN. INFO.:			US 1988-175970 A	19880331
			WO 1989-US1361 A	19890331

AB Luminides are a new class of drugs, defined as ABC, DABC, ADBC, or AB(D)C. A represents a functionality which is activatable by the environment and capable of transferring energy from its own excited state to the B functionality, which is an energy acceptor. Upon receiving energy from A, B achieves an excited state which relaxes through the heterolytic cleavage of the covalent bond of B with C, where C is a drug, which is released into the **intracellular compartment** where activation of A occurred. D serves as an electron transfer functionality which gains (loses) electrons from (to) the environment and donates (accepts) electrons to (from) A to activate it, so that the energy of excited A is transferred to B with release of C. MTL J-1 [5-phosphonoformate-1,5-di-[p-N-2-[N-(aminobutyl)-N-ethyisoluminol]-N-ethylaminophenyl]-1,5-bis-(p-N,N-dimethylaniline)-1,3-pentadiene] was **prepared** by known **methods.** Administration of MTL J-1 (10 μM total body weight

concentration) normalized spleen weight, more than did Foscarnet, in mice infected with Rauscher spleen focus-forming **virus**. The luminides might also include a biocompatible polymer and an immobilized enzyme.

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=> d que stat l51
L1             1 SEA FILE=REGISTRY ABB=ON  "LINOLEIC ACID"/CN
L13          894 SEA FILE=HCAPLUS ABB=ON  ?CELL?(W)?COMP? AND ?TRANSPLANT?
L14          105 SEA FILE=HCAPLUS ABB=ON  L13 AND (?REOVIRUS? OR ?REOVIRIDAE?
                 OR ?VIRUS?)
L34         6487 SEA CELL?(W) COMP? AND TRANSPLANT?
L35          718 SEA L34 AND (REOVIRUS? OR REOVIRID? OR VIRUS?)
L36            1 SEA L35 AND ONCOLYS?(3A) RAS?
L37           11 SEA L35 AND RAS?
L38            1 SEA L35 AND ONCOLYS?
L39           39 SEA L35 AND AUTOLOG?
L40          817 SEA L14 AND (MAMMAL? OR ANIMAL? OR BIRD? OR AVIAN? OR HUMAN?
                 OR SEROTYP?(W) 3 OR DEARING?(W) STRAIN?)
L41          690 DUP REMOV L40 (127 DUPLICATES REMOVED)
L42            1 SEA L41 AND (ANTI?(W) REOVIRUS? OR ANTIREOVIRUS?)
L43            1 SEA L41 AND IMMUN?(W) SYSTEM?(W) STIM?
L44           65 SEA L41 AND HEMATOP?(W) STEM?(W) CELL?
L45          105 SEA L36 OR L37 OR L38 OR L39 OR L42 OR L43 OR L44
L46           96 SEA L45 AND (BONE?(W) MARROW? OR BLOOD? OR TISSUE? OR ORGAN?
                 OR LIVER? OR KIDNEY? OR HEART? OR CORNEA? OR SKIN? OR LUNG? OR
                 PANCREAT? OR CULTUR?(W) CELL? OR SEMEN? OR EGG?)
L48            5 SEA L46 AND (FREEZ? OR STOR?)
L49            1 SEA L48 AND (L1 OR DMSO)
L50           96 SEA L46 OR L48 OR L49
L51            6 SEA L50 AND (METHOD? OR TECHNIQ? OR PROCED?)(3A)(PREP? OR
                 DEVEL? OR SYNTH?)


=> d ibib abs l51 1-6

L51  ANSWER 1 OF 6  WPIDS  COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER:     2003-606400 [57]   WPIDS
DOC. NO. CPI:         C2003-165103
TITLE:                Achieving endogenous development of lung,
                      gastrointestinal or skin cells in a recipient
                      from a bone marrow-derived stem cell
                      for treating e.g., HIV by transplanting the
                      bone marrow-derived stem cells into the
                      recipient.
DERWENT CLASS:        B04 D16
INVENTOR(S):          COLLECTOR, M I; KRAUSE, D S; SHARKIS, S J; THEISE, N D
PATENT ASSIGNEE(S):   (COLL-I) COLLECTOR M I; (KRAU-I) KRAUSE D S; (SHAR-I)
                      SHARKIS S J; (THEI-I) THEISE N D
COUNTRY COUNT:        1
PATENT INFORMATION:

     PATENT NO       KIND DATE     WEEK      LA   PG
     ---------------------------------------------
     US 2003095952   A1 20030522 (200357)*      18

APPLICATION DETAILS:

     PATENT NO      KIND                  APPLICATION         DATE
     ------------------------------------------------------------
     US 2003095952  A1 Provisional      US 2001-297927P      20010613
                                        US 2002-165533       20020607


PRIORITY APPLN. INFO: US 2001-297927P      20010613; US
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2002-165533 20020607

AN 2003-606400 [57] WPIDS

AB US2003095952 A UPAB: 20030906

NOVELTY - Achieving endogenous development of **lung**, gastrointestinal or **skin** cells in a recipient from a **bone marrow**-derived stem cell, is new.

DETAILED DESCRIPTION - Achieving endogenous development of **lung**, gastrointestinal or **skin** cells in a recipient from a **bone marrow**-derived stem **cell comprises**:

(a) providing a **bone marrow**-derived stem cells from a donor;

(b) providing a recipient having a defect in **lung**, gastrointestinal or epithelial cells;

(c) **transplanting** the **bone marrow** -derived stem cells into the recipient; and

(d) examining the **lung**, gastrointestinal or **skin** cells of the recipient to determine the presence or absence of endogenous development of **lung**, gastrointestinal or epithelial cells derived from the **bone marrow**-derived stem cells.

An INDEPENDENT CLAIM is also included for a **method** of achieving endogenous **development** of **lung**, gastrointestinal or **skin** cells in a recipient from a **bone marrow**-derived stem cell.

ACTIVITY - Anti-HIV; Virucide; Hepatotropic; Gastrointestinal. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The method is useful for achieving endogenous development of **lung**, gastrointestinal or **skin** cells in a recipient from a **bone marrow**-derived stem cell for treating Neimann Pick Disease, lactase deficiency, tyrosinemia, abetalipoproteinemia, glycogen **storage** diseases, alphalantitrypsin deficiency or cystic fibrosis, or viral infection, such as HIV, CMV, EBV or hepatitis C or B (claimed).

Dwg.0/4

L51 ANSWER 2 OF 6 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN

ACCESSION NUMBER: 2002-393966 [42] WPIDS

CROSS REFERENCE: 2002-292408 [33]

DOC. NO. CPI: C2002-110850

TITLE: Novel isolated **human** Neuropilin-Hy1 and Neuropilin-Hy2 polypeptides useful for treating neurodegenerative diseases e.g. Alzheimer's disease, and for diagnosing and mapping genetic neuronal defects.

DERWENT CLASS: B04 D16

INVENTOR(S): TANG, Y T

PATENT ASSIGNEE(S): (HYSE-N) HYSEQ INC

COUNTRY COUNT: 96

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002022815	A1	20020321	(200242)*	EN	152
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001089027	A	20020326	(200251)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002022815	A1	WO 2001-US28488	20010912
AU 2001089027	A	AU 2001-89027	20010912

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001089027	A	Based on	WO 2002022815

PRIORITY APPLN. INFO: US 2001-317902P 20010906; US 2000-659671 20000911

AN 2002-393966 [42] WPIDS

CR 2002-292408 [33]

AB WO 200222815 A UPAB: 20020812

NOVELTY - An isolated polypeptide (I) comprising a fully defined neuropilin-like polypeptide (Neuropilin-Hy1) sequence of 398 amino acids (S3) or a fully defined Neuropilin-Hy2 polypeptide sequence of 385 amino acids (S7) given in the specification, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polynucleotide (II) comprising a fully defined sequence of 1265, 1195,1907 or 1158 nucleotides as given in the specification;

(2) an isolated polynucleotide (III) encoding a polypeptide with biological activity, the polynucleotide having greater than about 99% sequence identity with (II); and

(3) a nucleic acid array (IV) comprising (II) attached to a surface.

ACTIVITY - Nootropic; neuroprotective; cytostatic; antianemic; vulnerary; antiulcer; antiparkinsonian; anticonvulsant; cerebroprotective; tranquilizer; anti-HIV; virucide; antibacterial; antiparasitic; protozoacide; immunosuppressive; dermatological; antiinflammatory; antirheumatic; antiarthritic; antithyroid; antidiabetic; ophthalmological.

No suitable data given.

MECHANISM OF ACTION - Modulator neuronal growth regenerative capacity; immune stimulator or suppressor; hematopoiesis regulator; gene therapy; modulator of (I).

USE - (IV) detects full-matches to (II) and also detects mismatches to (II) (claimed). The neuropilin-like polypeptides and polynucleotides are useful in modulating neuronal growth regenerative capacity, treating neurodegenerative diseases, diagnosing and mapping genetic neuronal defects and degenerative diseases like Alzheimer's disease. The neuropilin-like polypeptides and polynucleotides are also useful for treating learning and memory disorders. The polynucleotide and polypeptides are also useful for inducing angiogenesis, and neovascularization, as well as **organ** growth and development e.g. **heart** and other **tissues.**

Antagonists of neuropilin-like polypeptides are useful for treating cancers and other malignant diseases. The polynucleotides and polypeptides are also useful as markers for certain types of cancers. (I) is useful for generating antibodies that specifically bind the polypeptide, and are also useful as molecular weight markers and as food supplement. (I) is also useful for regulating stem cell growth factor activity, has hematopoiesis regulating activity, and is useful in supporting the growth and proliferation of megakaryocytes and consequently of platelets thereby allowing prevention or treatment of various platelet disorders such as

thrombocytopenia and/or in supporting growth and proliferation of **hematopoietic stem cells** which are capable of maturing to any and all of hematopoietic cells and therefore find therapeutic utility in various stem cell disorders those usually treated with **transplantation** such as a plastic anemia and paroxysmal nocturnal hemoglobinuria as well as in repopulating the stem **cell compartment** post irradiation/chemotherapy, etc., has **tissue** growth activity and is involved in nerve **tissue** growth or regeneration, in wound healing, **tissue** repair and replacement and in healing of bones, incisions and ulcers.

Compositions comprising (I) or (II) are useful for treating diseases of peripheral nervous system such as Alzheimer's, Parkinson's disease, Huntington's disease, amytrophic lateral sclerosis, and Shy-Drager syndrome, traumatic disorders, such as spinal cord disorders, head trauma and cerebrovascular diseases such as stroke, to promote better or faster closure of non-healing wounds, including without limitation pressure ulcers, ulcers associated with vascular insufficiency, surgical and traumatic wounds, etc. The polypeptides and polynucleotides also have chemotactic/chemokinetic activity, and are useful for cancer diagnosis and therapy.

The polypeptides are also useful for stimulating or suppressing activity of the immune system and therefore are useful for treating immune deficiencies and disorders. Therefore they are useful for treating immune deficiencies and disorders, infections by **human** immunodeficiency **virus** (HIV), hepatitis **viruses**, herpes **viruses**, mycobacteria, Leishmania spp., malaria spp., autoimmune disorders such as multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis, autoimmune pulmonary inflammation, autoimmune thyroiditis, insulin dependent diabetes mellitus, myasthenia gravis, graft-versus-host disease and autoimmune inflammatory eye disease.

(II) are also useful as hybridization probes, as oligomers or primers for polymerase chain reaction (PCR), in computer readable media, for chromosome and gene mapping, recombinant production of proteins and in the generation of antisense DNA or RNA or their chemical analogs. (II) is useful in gene therapy techniques. The polypeptides are useful in in vitro or in vivo inhibition of cellular function, and for identifying compounds that modulate the expression or activity of (I) or (II). (I) and (II) are also useful for evaluating the efficacy of drugs and monitoring the progress of patients involved in clinical trials for the treatment of disorders.

(I) and (II) have research uses and utilities e.g., the polynucleotides are useful for expressing recombinant protein for analysis, characterization or therapeutic use, as markers for **tissues** in which the corresponding protein is preferentially expressed as chromosome markers or tags and the polypeptides are useful for making antibodies that are specifically reactive with (I). Modulators of (I) expression or activity are useful for treating the above mentioned conditions.

Dwg.0/17

L51 ANSWER 3 OF 6 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN

ACCESSION NUMBER: 2002-055133 [07] WPIDS
CROSS REFERENCE: 2003-381586 [36]
DOC. NO. CPI: C2002-015672
TITLE: Purifying complexes comprising GRP94 proteins, useful for treating a disorder associated with ischemia/reperfusion.
DERWENT CLASS: B04 D16
INVENTOR(S): NICCHITTA, C V; REED, R C; ROSSER, M F N; WASSENBERG, J J; GEWIRTH, D T
PATENT ASSIGNEE(S): (UYDU-N) UNIV DUKE; (GEWI-I) GEWIRTH D T; (NICC-I)

NICCHITTA C V; (REED-I) REED R C; (ROSS-I) ROSSER M F N; (WASS-I) WASSENBERG J J

COUNTRY COUNT: 96

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001072779	A1	20011004	(200207)*	EN	169
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001047759	A	20011008	(200208)		
US 2002160496	A1	20021031	(200274)		
EP 1265913	A1	20021218	(200301)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
US 2003054996	A1	20030320	(200323)		
JP 2003528886	W	20030930	(200365)		178

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2001072779	A1		WO 2001-US9512	20010326
AU 2001047759	A		AU 2001-47759	20010326
US 2002160496	A1	Provisional	US 2000-192118P	20000324
		CIP of	WO 2001-US9512	20010326
			US 2001-968436	20011001
EP 1265913	A1		EP 2001-920734	20010326
			WO 2001-US9512	20010326
US 2003054996	A1	Provisional	US 2000-192118P	20000324
		Cont of	WO 2001-US9512	20010326
			US 2002-210333	20020801
JP 2003528886	W		JP 2001-571710	20010326
			WO 2001-US9512	20010326

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001047759	A	Based on	WO 2001072779
EP 1265913	A1	Based on	WO 2001072779
JP 2003528886	W	Based on	WO 2001072779

PRIORITY APPLN. INFO: US 2000-192118P 20000324; US 2001-968436 20011001; US 2002-210333 20020801

AN 2002-055133 [07] WPIDS

CR 2003-381586 [36]

AB WO 200172779 A UPAB: 20031009

NOVELTY - Purifying a complex of a GRP94 protein, comprising contacting a complex with the GRP94 protein to bind it an agent immobilized on a solid phase support, collecting the remaining sample, and eluting the complex from the solid phase support, is new.

DETAILED DESCRIPTION - Purifying a complex of a GRP94 protein, comprising:

(a) contacting a complex comprising a GRP94 protein with a binding

agent that preferentially binds GRP94, the binding agent immobilized to a solid phase support, to immobilize the complex to the solid phase support;

(b) collecting the remaining sample; and

(c) eluting the complex from the solid phase support to give purified complex in the eluate.

INDEPENDENT CLAIMS are also included for the following:

(1) isolating an antigenic molecule, associated with a GRP94 complex, comprising:

(a) contacting a complex comprising a GRP94 protein with a binding agent that preferentially binds GRP94, the binding agent immobilized to a solid phase support, to immobilize the complex to the solid phase support;

(b) collecting the remaining sample;

(c) eluting the complex from the solid phase support to give purified complex in the eluate; and

(d) isolating the antigenic molecule from the eluate;

(2) a product produced by either of the novel method, or the method of (1);

(3) detecting a complex comprising GRP94 in a sample suspected of containing a complex comprising GRP94, comprising:

(a) contacting the sample with a binding agent that preferentially binds GRP94 under conditions favorable to binding a complex comprising GRP94 to the binding substance to form a second complex; and

(b) detecting the second complex via a label conjugated to the binding substance or via a labeled reagent that specifically binds to the second complex subsequent to its formation;

(4) a kit for detecting, isolating or purifying a complex comprising GRP94 or an antigenic molecule associated with a complex comprising GRP94, the kit comprising:

(a) a binding agent that preferentially binds GRP94 contained in a first container; and

(b) an elution buffer for use in eluting a complex comprising GRP94 from the binding agent, the elution buffer contained in a second container;

(5) screening a candidate substance for an ability to modulate GRP94 biological activity, comprising:

(a) establishing a test sample comprising a GRP94 protein and a ligand for a GRP94 protein;

(b) administering a candidate substance to the test sample; and

(c) measuring the effect of the candidate substance on binding of the GRP94 protein and the ligand in the test sample;

(6) screening a candidate substance as an activator (or inhibitor) of the biological activity of a Hsp90 protein, comprising:

(a) establishing a test sample comprising a Hsp90 protein and a candidate substance;

(b) administering 1,8 -anilinonaphthalenesulfonate (8-ANS) to the test sample;

(c) detecting a fluorescence signal produced by the 8-ANS; and

(d) identifying the candidate substance as an activator (or inhibitor) of the biological activity of a Hsp90 protein based upon an amount of fluorescence signal produced by the 8-ANS as compared to a control sample;

(7) modulating the biological activity of a Hsp90 protein, comprising contacting a Hsp90 protein with an effective amount of a Hsp90 protein activity-modulating substance to thereby modulate the biological activity;

(8) treating a subject from a disorder where modulation of the biological activity of a GRP94 protein is desirable, comprising administering to the subject an effective amount of a GRP94 protein modulator;

(9) altering a subsequent cellular response to an ischemic condition at a tissue location in a subject, comprising treating the cells at the

tissue location with a GRP94 protein modulator

(10) preparing an immunogenic composition for inducing an immune response in a vertebrate subject, comprising:

(a) harvesting from a eukaryotic cell an immunogenic complex comprising a Hsp90 protein non-covalently bound to an antigenic molecule, the complex when administered to the vertebrate subject being operative at initiating an immune response in the vertebrate subject, wherein the eukaryotic cell has been treated with an activating ligand of a Hsp90 protein; and

(b) combining the complex with a pharmaceutically acceptable carrier;

(11) preparing an immunogenic composition for inducing an immune response in a vertebrate subject, comprising:

(a) reconstituting in vitro an antigenic molecule and a Hsp90 protein molecule in the presence of a modulator of the biological activity of a Hsp90 protein to thereby produce an immunogenic complex comprising a Hsp90 protein non-covalently bound to an antigenic molecule, the complex when administered to the vertebrate subject being operative at initiating an immune response in the vertebrate subject; and

(b) combining the complex with a pharmaceutically acceptable carrier;

(12) preparing an immunogenic composition for inducing an immune response in a vertebrate subject, comprising:

(a) sensitizing one or more antigen presenting cells in vitro with a complex comprising a Hsp90 protein non-covalently bound to an antigenic molecule and with an activating ligand of a Hsp90 protein; and

(b) combining the one or more sensitized antigen presenting cells with a pharmaceutically acceptable carrier; and

(13) a product produced by one of the methods of (10)-(12).

ACTIVITY - Cardiant; Vasodilator; Hypertensive; Hyperglycemic; Anticonvulsant; Neuroprotective; Nootropic; Neuroleptic; Anxiolytic.

No biological data is given.

MECHANISM OF ACTION - GRP94 modulator.

USE - The method of (8) can be used to treat a disorder associated with ischemia/reperfusion as a result of cardiac arrest, asystole and sustained ventricular arrhythmias, cardiac surgery, cardiopulmonary bypass surgery, organ transplantation, spinal cord injury, head trauma, stroke, thromboembolic stroke, hemorrhagic stroke, cerebral vasospasm, hypotension, hypoglycemia, status eliepticus, an epileptic seizure, anxiety, schizophrenia, a neurodegenerative disorder, Alzheimer's disease, Huntington's disease, amyotrophic lateral sclerosis (ALS), or neonatal stress (claimed).

ADVANTAGE - None given.

Dwg.0/14

L51 ANSWER 4 OF 6 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN

ACCESSION NUMBER: 2002-049344 [06] WPIDS

CROSS REFERENCE: 2002-011412 [01]

DOC. NO. NON-CPI: N2002-036482

DOC. NO. CPI: C2002-013890

TITLE: Removing **ras**-mediated neoplastic cells from a **cellular composition** by contacting the composition with **reovirus** which results in **oncolysis** of neoplastic cells, useful for increasing efficacy of **hematopoietic stem cell transplantation.**

DERWENT CLASS: B04 D16 P34

INVENTOR(S): COFFEY, M C; MORRIS, D; THOMPSON, B G

PATENT ASSIGNEE(S): (ONCO-N) ONCOLYTICS BIOTECH INC

COUNTRY COUNT: 95

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001083711	A2	20011108	(200206)*	EN	41
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001058086	A	20011112	(200222)		
EP 1278824	A2	20030129	(200310)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
BR 2001010474	A	20030401	(200327)		
JP 2003531606	W	20031028	(200373)		44
MX 2002010744	A1	20030501	(200415)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001083711	A2	WO 2001-CA620	20010502
AU 2001058086	A	AU 2001-58086	20010502
EP 1278824	A2	EP 2001-931251	20010502
		WO 2001-CA620	20010502
BR 2001010474	A	BR 2001-10474	20010502
		WO 2001-CA620	20010502
JP 2003531606	W	JP 2001-580320	20010502
		WO 2001-CA620	20010502
MX 2002010744	A1	WO 2001-CA620	20010502
		MX 2002-10744	20021031

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001058086	A	Based on	WO 2001083711
EP 1278824	A2	Based on	WO 2001083711
BR 2001010474	A	Based on	WO 2001083711
JP 2003531606	W	Based on	WO 2001083711
MX 2002010744	A1	Based on	WO 2001083711

PRIORITY APPLN. INFO: US 2001-268054P 20010213; US 2000-201990P 20000503; US 2000-205389P 20000519

AN 2002-049344 [06] WPIDS

CR 2002-011412 [01]

AB WO 200183711 A UPAB: 20040302

NOVELTY - A new method (M1) to remove **ras**-mediated neoplastic cells from a **cellular composition** suspected of containing such neoplastic **cells**, **comprises** contacting the **cellular composition** with **reovirus** under conditions which results in **oncolysis** of the **ras**-mediated neoplastic cells.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a **method** (M2)of **preparing** a **cellular composition** for **transplantation** into a recipient, comprising selecting a **cellular composition** for **transplantation** and contacting the composition with a

reovirus under conditions which result in **oncolysis** of **ras**-meditated neoplastic cells;

(2) a method (M3) of reducing a risk of recurrence of tumor due to **transplantation** of **autologous hematopoietic stem cell** suspected of containing **ras**-mediated neoplastic **cells comprising** harvesting from a subject to receive the **transplant** a **cellular composition** which comprises **hematopoietic stem cells**, contacting the **cellular composition** with a **reovirus** under conditions which result in **oncolysis** of **ras**-mediated neoplastic cells, and introducing the **reovirus**-treated composition back into the subject; and

(3) a **cellular composition**, comprising **hematopoietic stem cells**, prepared by M1.

ACTIVITY - Cytostatic.

No biological data given.

MECHANISM OF ACTION - The **reovirus** causes the **oncolysis** of the **ras**-mediated neoplastic cells.

No biological data given.

USE - The method is useful for treating stem cell containing autographs with **reovirus** prior to **transplantation** to remove contaminating or spontaneous **ras**-mediated neoplastic cells. This increases the efficacy of the high dose chemotherapy/ **autologous hematopoietic stem cell transplantation** treatment of Hodgkin's disease, multiple myeloma brain tumors and breast tumors.

The **cellular composition** comprises a **tissue**, an **organ** or any portion of a **tissue** or an **organ.** Alternatively, the **cellular composition** comprises **cultured cells**, **semen** or **eggs**

The composition is used in **transplantation** (claimed).

Dwg.0/4

L51 ANSWER 5 OF 6 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-476199 [51] WPIDS
CROSS REFERENCE: 2001-442253 [47]; 2001-442255 [47]; 2001-451890 [48]; 2001-451908 [48]; 2001-451909 [48]; 2001-451912 [48]; 2001-451938 [48]; 2001-451939 [48]; 2001-457603 [49]; 2001-457740 [49]; 2001-465363 [50]; 2001-465571 [50]; 2001-465578 [50]; 2001-465705 [50]; 2001-476114 [51]; 2001-476164 [51]; 2001-476197 [51]; 2001-476198 [51]; 2001-476282 [51]; 2001-476283 [51]; 2001-483140 [52]; 2001-483233 [52]; 2001-488707 [53]; 2001-488788 [53]; 2001-488875 [53]; 2001-488895 [53]; 2001-496929 [54]; 2001-496930 [54]; 2001-496931 [54]; 2001-496932 [54]; 2001-514838 [56]; 2001-522358 [57]; 2001-565565 [63]; 2001-582152 [65]; 2001-582153 [65]; 2001-589862 [66]; 2001-589934 [66]; 2001-607699 [69]; 2001-611724 [70]; 2001-611725 [70]; 2001-626375 [72]; 2001-626426 [72]; 2001-626432 [72]; 2001-626527 [72]; 2001-639362 [73]; 2002-010428 [01]; 2002-025688 [03]; 2002-062370 [08]; 2002-280918 [32]; 2002-426278 [45]; 2002-575369 [61]; 2002-590824 [63]; 2002-674924 [72]; 2003-018710 [01]; 2003-028924 [02]; 2003-110596 [10]; 2003-174164 [17]; 2003-313249 [30]; 2003-456302 [43]; 2003-678194 [64]; 2003-679633 [64]; 2003-697229 [66]; 2003-697230 [66]; 2003-697231 [66]; 2003-810980 [76]; 2003-829799 [77]; 2003-851723 [79]; 2003-852227 [79]; 2004-061257 [06];

2004-089285 [09]; 2004-143291 [14]; 2004-167906 [16]; 2004-169496 [16]
DOC. NO. CPI: C2001-142863
TITLE: Novel carcinoembryonic antigen-like protein, useful for treating breast, prostate and colon cancers, inflammatory and autoimmune disorders, as immunosuppresant, as decoy receptor in bacterial and viral infections.
DERWENT CLASS: B04 D16
INVENTOR(S): ARTERBURN, M C; BOYLE, B J; DRMANAC, R A; KUO, C; LIU, C; TANG, Y T
PATENT ASSIGNEE(S): (HYSE-N) HYSEQ INC
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001055337	A2	20010802	(200151)*	EN	131
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001036553	A	20010807	(200174)		
EP 1276902	A2	20030122	(200308)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
JP 2004500078	W	20040108	(200410)		223

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001055337	A2	WO 2001-US2614	20010125
AU 2001036553	A	AU 2001-36553	20010125
EP 1276902	A2	EP 2001-908711	20010125
		WO 2001-US2614	20010125
JP 2004500078	W	JP 2001-554369	20010125
		WO 2001-US2614	20010125

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001036553	A	Based on	WO 2001055337
EP 1276902	A2	Based on	WO 2001055337
JP 2004500078	W	Based on	WO 2001055337

PRIORITY APPLN. INFO: US 2000-665533 20000919; US 2000-491404 20000125
AN 2001-476199 [51] WPIDS
CR 2001-442253 [47]; 2001-442255 [47]; 2001-451890 [48]; 2001-451908 [48]; 2001-451909 [48]; 2001-451912 [48]; 2001-451938 [48]; 2001-451939 [48]; 2001-457603 [49]; 2001-457740 [49]; 2001-465363 [50]; 2001-465571 [50]; 2001-465578 [50]; 2001-465705 [50]; 2001-476114 [51]; 2001-476164 [51]; 2001-476197 [51]; 2001-476198 [51]; 2001-476282 [51]; 2001-476283 [51]; 2001-483140 [52]; 2001-483233 [52]; 2001-488707 [53]; 2001-488788 [53]; 2001-488875 [53]; 2001-488895 [53]; 2001-496929 [54]; 2001-496930 [54]; 2001-496931 [54]; 2001-496932 [54]; 2001-514838 [56]; 2001-522358 [57]; 2001-565565 [63]; 2001-582152 [65]; 2001-582153 [65]; 2001-589862 [66];

2001-589934 [66]; 2001-607699 [69]; 2001-611724 [70]; 2001-611725 [70]; 2001-626375 [72]; 2001-626426 [72]; 2001-626432 [72]; 2001-626527 [72]; 2001-639362 [73]; 2002-010428 [01]; 2002-025688 [03]; 2002-062370 [08]; 2002-280918 [32]; 2002-426278 [45]; 2002-575369 [61]; 2002-590824 [63]; 2002-674924 [72]; 2003-018710 [01]; 2003-028924 [02]; 2003-110596 [10]; 2003-174164 [17]; 2003-313249 [30]; 2003-456302 [43]; 2003-678194 [64]; 2003-679633 [64]; 2003-697229 [66]; 2003-697230 [66]; 2003-697231 [66]; 2003-810980 [76]; 2003-829799 [77]; 2003-851723 [79]; 2003-852227 [79]; 2004-061257 [06]; 2004-089285 [09]; 2004-143291 [14]; 2004-167906 [16]; 2004-169496 [16]

AB WO 200155337 A UPAB: 20040326

NOVELTY - An isolated polypeptide (carcinoembryonic antigen (CEA)-like protein) (I) comprising an amino acid sequence which is at least 80% identical to a fully defined sequence of 425 (S4), 45, 45, 20, 405, 45 (S6-S10) amino acids as given in the specification, a mature protein or its extracellular portion or active domain, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a polypeptide (Ia) having CEA-like activity comprising 10 consecutive amino acids of (S4) and (S6)-(S10);

(2) an isolated polynucleotide (II) comprising a fully defined sequence of 416 (S2), 1557 (S3) or 1278 (S5) nucleotides as given in the specification, its translated protein coding portion, the mature protein coding portion, the extracellular portion, or active domain;

(3) an isolated polynucleotide encoding a polypeptide with biological activity, which hybridizes to the complement of (II) under stringent hybridization conditions;

(4) an isolated polynucleotide encoding a polypeptide with biological activity, where the polynucleotide has greater than 90% sequence identity with (II);

(5) an isolated polynucleotide which comprises the complement of (II);

(6) a vector comprising (II);

(7) an expression vector comprising (II);

(8) a host cell (III) genetically engineered to express (II);

(9) a composition comprising (I) and a carrier;

(10) a polynucleotide encoding (I) or (Ia);

(11) an antibody specific for (I);

(12) detecting (M1) (II) in a sample involves, contacting the sample with a compound that binds to and forms a complex with (II) to form a complex and detecting the complex, so that if a complex is detected, (II) is detected. The method alternately (M2) involves contacting the sample under stringent hybridization conditions with nucleic acid primers that anneal to (II), amplifying a product comprising at least a portion of (II) and detecting the product and thereby (II) in the sample;

(13) detecting (I) in a sample involves contacting the sample with a compound that binds to and forms a complex with (I) to form a complex and detecting the complex, so that if a complex is detected, (I) is detected;

(14) identifying a compound that binds to (I) involves contacting the compound with the polypeptide to form a polypeptide/compound complex and detecting the complex, so that if the polypeptide/compound complex is detected, a compound that binds to (I) is identified;

(15) the method alternately involves contacting the compound with (I), in a cell, to form a polypeptide/compound complex, where the complex drives expression of a reporter gene sequence in the cell and detecting the complex by detecting reporter gene sequence expression, so that if the polypeptide/compound complex is detected, a compound that binds to (I) is identified;

(16) preparation of (I);

(17) a kit comprising (I);

(18) a nucleic acid array (IV) comprising (II) or a unique segment of (II) attached to the surface;

(19) treating a subject in need of enhanced activity or expression of (I) involves administering an agonist of (I), (I) or a polynucleotide encoding (I) under conditions such that the polypeptide is produced, and a carrier; and

(20) treating a subject in need to inhibit activity or expression of (I) involves administering an antagonist of (I), a polypeptide that competes with (I) for its ligand or a polynucleotide that inhibits the expression of a nucleotide sequence encoding (I), and a carrier.

ACTIVITY - Cytostatic; antiinflammatory; immunosuppressive; antianemic; vulnerary; osteopathic; antiarthritic; antiulcer; nootropic; neuroprotective; cerebroprotective; immunostimulant; antirheumatoid; antithyroid; virucide; contraceptive; antiinfertility; hemostatic; thrombolytic; anticoagulant; antibacterial; antiparkinsonian; vasotropic.

No supporting data is given.

MECHANISM OF ACTION - CEA-like protein expression or activity modulator; antisense therapy or gene therapy; cell development, proliferation, growth, differentiation, survival, regeneration, immune responses modulator.

USE - (II) is useful as hybridization probes, oligomers or primers, in computer readable media, chromosome and gene mapping for recombinant production of (I) and in generation of antisense DNA or RNA, their chemical analogs, etc. They are also useful as diagnostics. (II) can be used to induce immune responses. (I) is useful for generating antibodies, as molecular weight markers and as a food supplement. (I) can be used for in vitro biding assays to identify molecules which bind to the polypeptide.

(I) and (II) can be used for treating breast, prostate, colon and other cancers, disorders relating to inflammation and autoimmunity, as immunosuppressant in **organ transplantations**, as a decoy receptor in bacterial and viral infections. Detecting (I) or (II) is used as part of prognostic or diagnostic evaluation of disorders and for identifying subjects exhibiting predisposition to such conditions.

The novel polynucleotides can be used to express recombinant protein for analysis, characterization or therapeutic use, as markers for **tissues** in which the corresponding protein is preferentially expressed (either constitutively or at a particular stage of **tissue** differentiation or development or in disease states), as molecular weight markers on Southern gels, as chromosome markers or tags (when labeled) to identify chromosomes or to map related gene positions, to compare with endogenous DNA sequences in patients to identify potential genetic disorders, as probes to hybridize and thus discover novel, related DNA sequences, as source of information to derive polymerase chain reaction (PCR) primers for genetic fingerprinting, as a probe to subtract-out known sequences in the process of discovering other novel polynucleotides, for selecting and making oligomers for attachment to a gene chip or other support, including for examination of expression patterns, to raise anti-DNA antibodies or elicit another immune response.

The novel proteins can similarly be used in assays to determine biological activity, including in a panel of multiple proteins for high throughput screening, to raise antibodies or to elicit another immune response, as a reagent (including the labeled reagent) in assays designed to quantitatively determine levels of the protein (or its receptor) in biological fluids, as markers for **tissues** in which the corresponding protein is preferentially expressed (either constitutively or at a particular stage of **tissue** differentiation or development or in a disease state). Proteins involved in these binding interactions can also be used to screen for peptide or small molecular inhibitors or agonists of the binding reactions. The proteins can also be

used for making antibody substances that are specifically immunoreactive with CEA-like proteins.
Dwg.0/2

L51 ANSWER 6 OF 6 WPIDS COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-374265 [39] WPIDS
DOC. NO. CPI: C2001-114294
TITLE: Pretreating **animal** for inducing tolerance to gene transfer products by treating **animal** with **hematopoietic stem cells** transduced with vector or polynucleotide, which is to be introduced into **animal** through gene therapy.
DERWENT CLASS: B04 D16
INVENTOR(S): ANDERSSON, G K
PATENT ASSIGNEE(S): (BIOT-N) BIOTRANSPLANT INC
COUNTRY COUNT: 93
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001025398	A2	20010412	(200139)*	EN	69
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW					
AU 2000077406	A	20010510	(200143)		
JP 2003531816	W	20031028	(200373)		60

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001025398	A2	WO 2000-US26946	20000929
AU 2000077406	A	AU 2000-77406	20000929
JP 2003531816	W	WO 2000-US26946	20000929
		JP 2001-528553	20000929

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000077406	A	Based on	WO 2001025398
JP 2003531816	W	Based on	WO 2001025398

PRIORITY APPLN. INFO: US 1999-157233P 19991001
AN 2001-374265 [39] WPIDS
AB WO 200125398 A UPAB: 20010716
NOVELTY - Pretreating an **animal** that is to receive one of a vector (I) encoding a therapeutic polypeptide or recombinant **cells comprising** (I) or a polynucleotide (II) encoding the therapeutic polypeptide involves treating the **animal** with **hematopoietic stem cells** (HSC) transduced with (I) or (II).

ACTIVITY - Antianemic; immunostimulant; hemostatic; antilipemic; immunosuppressive; cytostatic.

MECHANISM OF ACTION - Gene therapy. No supporting data is given.

USE - Pretreating an **animal** that is to receive one of (I) encoding a therapeutic polypeptide to alleviate a genetic deficiency

disease or recombinant **cells comprising** (I) or a (II) encoding the therapeutic polypeptide. The genetic deficiency disease which is alleviated by the gene product encoded by (I) is cystic fibrosis, muscular dystrophy, hemophilia A, hemophilia B, familial hypercholesterolemia, hemoglobinopathies, thalassemia, sickle cell anemia, Gaucher's disease, alpha 1-antitrypsin deficiency, inherited emphysema, chronic granulomatous disease, Fanconi's anemia, and immunodeficiency disease. The therapeutic gene product also acts to reduce a detrimental immune response such as an autoimmune disease or an atopic disease. Also the therapeutic gene acts to alleviate or prevent cancer in a patient afflicted with or is at risk for developing cancer. In this case the pretreatment method involves introducing into the **animal**, a vector (e.g. adenoviral or retroviral vector) that transduces cancer cells and which contains a gene (Herpes simplex **virus** thymidine kinase (HSV-TK) whose gene product will sensitize the cancer cells to one or more cytotoxic agents e.g. gancyclovir (claimed). The method is useful for alleviating or ameliorating adverse immune response and inducing immunological tolerance in an **animal** receiving genetically different cells or gene therapy vectors. The method inhibits adverse immune responses to **transplantation** through **transplantation** of **organs** or as a result of gene therapy. The **methods develop** immunological tolerance in gene therapy, utilizing the host's ability to mount an immune response against neoantigens in a beneficial manner.

ADVANTAGE - The methods are suitable for inducing immunological tolerance in an **animal.** Severe problems associated with immune responses directed against transgene encoded proteins are effectively eliminated by this method.

Dwg.0/1